Diagnostic and Initial Treatment Of Occupational Asthma

I. Background:

A. Asthma is an airways disease of the lungs characterized by the following:

1. Airway inflammation
2. Increased airway responsiveness to a variety of stimuli; and
3. Airway obstruction that is partially or completely reversible, either spontaneously or with treatment.

The two essential *clinical* elements for the diagnosis of asthma are airways obstruction which is partially or totally reversible with treatment, and/or airways hyperreactivity. *Occupational asthma* is asthma that has its onset in association with workplace exposure(s). *Occupationally – aggravated asthma* is asthma that is aggravated by workplace exposure(s).

B. Causative agents are classified as sensitizers (including but not limited to the appended list) or irritants. Sensitizers cause inflammation through one or more immunologic mechanisms, whereas irritants directly inflame the airway. Occupational environments are often complex, and it may be difficult to identify a single specific causal agent.

C. A delay in diagnosis resulting in continued exposure of the worker to even minute amounts of sensitizers can lead to permanent and irreversible airways disease or *death.*

D. An acute high level inhalation exposure to an irritant may result in a permanent asthmatic condition known as Reactive Airways Dysfunction Syndrome (RADS).

E. This guideline is meant to cover the majority of tests and treatments that may be used to diagnose and initially stabilize occupational and occupationally-aggravated asthma. **This guideline does not include parameters of care for long term management of either occupational or occupationally-aggravated asthma.** It is expected that approximately 10% of cases will fall outside this guideline and require review on a case-by-case basis.

II. Criteria for Diagnosis:

A. Diagnosis of Occupational Asthma

1. Diagnosis of asthma within these guidelines by a medical doctor, using the appended algorithm.
2. Historical association between the onset of asthma and work,
AND
3. At least one of the following criteria:

a. Documentation (see Occupational History, Section III.B) of workplace exposure to a category of agents or processes associated with asthma;
b. Work-related change in FEV1 or in peak expiratory flow (PEF);
c. Onset of respiratory signs and/or symptoms within hours after an acute high level occupational inhalation exposure to an irritant (RADS)

B. Diagnosis of Occupationally-Aggravated Asthma: There must be a history of asthma prior to the occupational exposure in question. Other diagnostic criteria are the same as for new onset occupational asthma.

III. Medical Diagnosis and Initial Stabilization:

Physician Visits Allowed. The number of physician visits needed to diagnose and stabilize cases of occupational and occupationally-aggravated asthma is likely to vary from patient to patient. Physicians must use their judgment to determine the number of physician visits necessary for diagnosis and initial stabilization.

IV. Establishing the Diagnosis:

A. Medical History:

1. Characteristic symptoms: wheeze, cough, chest tightness, shortness of breath.
2. Past respiratory history: prior diagnosis of asthma, allergies, eczema, rhinitis, bronchitis, sinusitis, hayfever, chest colds, and respiratory symptoms upon exertion, exposure to minor irritants, or exposure to cold air.
3. Review of systems: history of other diseases with symptoms that could mimic or precipitate asthma; e.g., cardiovascular disease with left ventricular dysfunction; gastroesophageal reflux.
4. Family history: asthma, atopy.

5. Smoking history: average # packs of cigarettes per day x # years smoked (pack years of smoking).
6. List of current medications.
7. Home, hobby, and environmental exposure history to exclude other causal or contributing factors.

B. Occupational History:

1. Description of the patient's work tasks, exposures and related processes, both past and present.
2. Effect(s) of workplace exposures on respiratory symptoms, with emphasis on temporal associations. Note whether symptoms change on weekends and/or vacation.
3. Documentation of workplace exposures where possible: e.g., Material Safety Data Sheets (MSDS); employer records; industrial hygiene monitoring data from government agencies or private consultants.
4. Where data for characterizing exposures is inadequate, worksite evaluation by an appropriate health care provider or industrial hygienist may be necessary and is encouraged.

C. Physical Examination:

1. Examination of head for rhinitis, nasal polyps, conjunctivitis, and sinusitis.
2. Chest percussion and auscultation.
3. Cardiovascular exam to rule out cardiogenic explanation for respiratory symptoms.
4. Skin exam for atopic dermatitis.

D. Diagnostic Tests Allowed:

1. A total of 11 spirometry *studies* is allowed. For purposes of this guideline, each *study* shall consist of a minimum of 3 and a maximum of 8 *maneuvers*, with at least the initial study pre- and post-inhaled bronchodilator.

    a. Up to 2 follow-up spirometry studies will be allowed to establish a diagnosis of asthma.
    b. Up to 8 pre- and post-shift spirometry studies will be allowed at the beginning and end of each work week for 2 weeks.
    c. When PEF diary and spirometric monitoring are equivocal, a longer absence from work may be needed to establish or rule out the diagnosis, with

(i) 1 repeat spirometry study allowed at the beginning of the absence from work and 1 repeat spirometry study allowed at the end of the absence from work, and
(ii) the PEF diary monitoring repeated.

2. One Non-Specific Inhalation Challenge Test Allowed:
If there is no significant improvement in FEV1 in response to inhaled bronchodilator, and *if* the existence of airways hyperreactivity remains in question (see appended algorithm), but *only* when:

a. Performed in a Hospital-based Outpatient Setting,
b. Consistent with this guideline's Appended Algorithm, and
c. Under Supervision of a medical doctor experienced in this type of procedure.

3. Ten Specific Skin Tests with relevant antigens allowed, but *only* when:

a. Performed by a Medical Doctor Experienced in this type of Procedure and,
b. In a hospital-based outpatient setting.

WARNING: SKIN TESTS ARE NON-EMERGENT PROCEDURES, WITH SIGNIFICANT RISK OF SEVERE REACTION, INCLUDING DEATH.

4. Chest radiograph – 1 postero-anterior and 1 lateral view allowed.
5. Latex and laboratory animal dander RAST test(s) for specific work-related exposure – 1 allowed for each antigen.

V. Initial Treatment Program:

A. Prevention of further exposure to causal or precipitating agent(s):

1. When caused by a sensitizing agent, all further exposure to the causal agent must be eliminated because of the increased risk for irreversible airways obstruction, severe bronchospasm and/or *death*. A statement of the physician's discussion of these and other risks with the patient must be documented in the medical record.
2. When caused by an irritant, elimination of exposure is desirable but significant reduction of exposure may be sufficient. When elimination of exposure is not possible, alternative approaches may

include, in order of preference:

a. Engineering controls such as local exhaust ventilation

b. Appropriate use of respiratory protection provided by the employer

B. Where these approaches fail and the clinical condition warrants, removal of the workers from the workplace may be necessary.

C. Medications:

1. Medications should only be used in conjunction with prevention of further exposure as outlined in Section V. A. above.

2. Spirometric testing is allowed as needed to monitor effectiveness of therapy, not to exceed a maximum of 11 spirometry studies allowed in Section IV. D. above. Due to its unique nature, Occupational Asthma often requires a more aggressive therapeutic approach than Non-Occupational Asthma. The recommended therapeutic approach is as follows:

a. Step 1: Rapid-onset *B*-agonist as needed for control of symptoms of asthma occurring less than three times per week. If this fails, then:
b. Step 2: Inhaled low-to-medium dose corticosteroids to treat underlying inflammation, combined with a rapid-onset inhaled *B*-agonist as needed to control symptoms of asthma. If this fails, then:
c. Step 3: Increase inhaled corticosteroids to high dose, plus long-acting inhaled *B*-agonist, and/or oral *B*-agonist and/or theophylline with continued use of rapid-onset inhaled *B*-agonist as needed to control symptoms of asthma. If this fails, then:
d. Step 4: Add an oral corticosteroid.

D. Patient Education (The following shall be discussed with the patient at the initial physician visit and repeated thereafter as necessary):

1. Key points about signs and symptoms of asthma and characteristic airway changes in asthma.
2. Asthma triggers and how to avoid them.
3. How medications work and their potential adverse effects; instruction and demonstration in the correct use of all medications (e.g., proper use of MDIs).
4. Techniques of monitoring status of asthma, such as PEF readings.
5. Indications for emergency care.

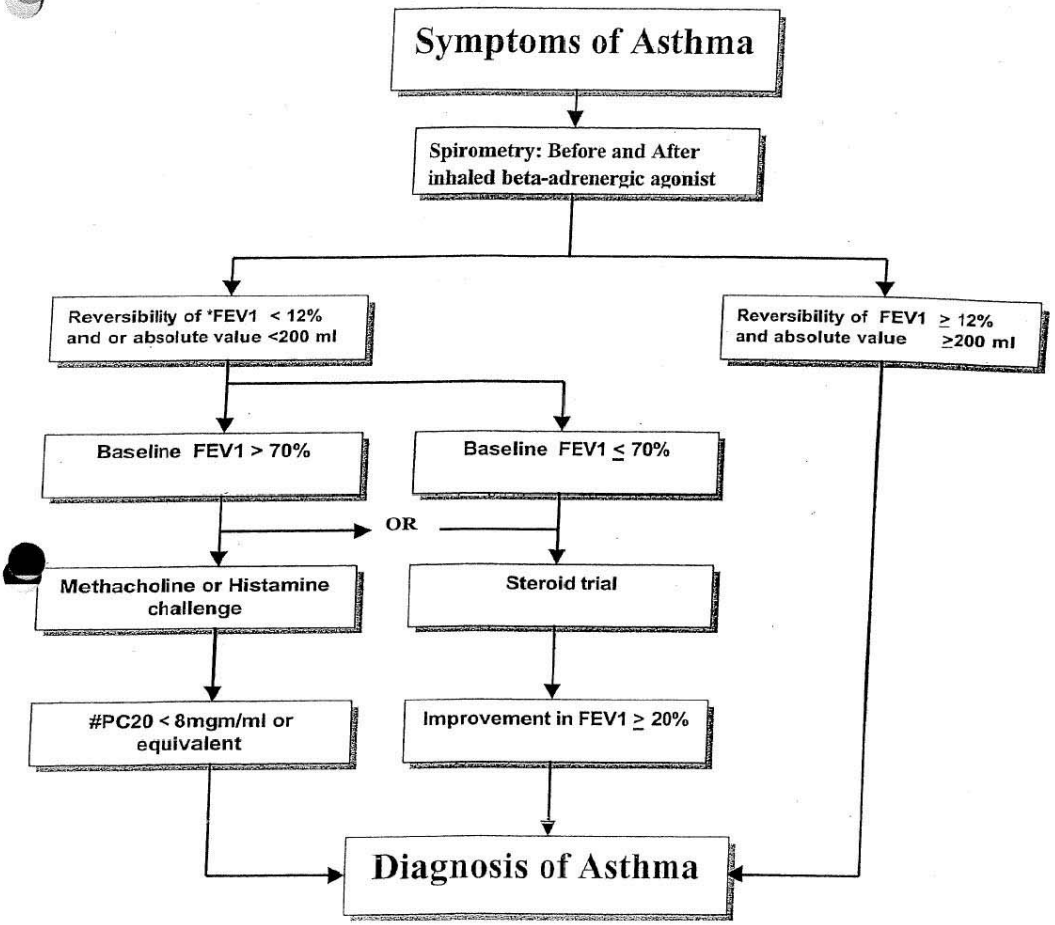
VI. Discharge Plan:

A. Future medical care will depend upon the outcome of initial medical management. This guideline is meant to address only the diagnosis and initial stabilization of occupational and occupationally-aggravated asthma.

B. If causal or aggravating exposure is eliminated or reduced and asthma symptoms resolve without medication, no further medical management is needed. If symptoms have resolved with medication, a period of medical follow-up will be needed to determine the necessity for continued medication and to establish an effective maintenance regimen. Practitioners should consult other guidelines, practice parameters and/or standards of care for guidance in the long-term management of persistent symptoms of asthma.

Protocol History:
Passed: April 20, 2004
Effective: May 11, 2004

DIAGNOSIS OF ASTHMA ALGORITHM



FEV1 = Forced Expiratory Volume in one second
#PC20 = Provocative concentration to cause a 20% decline in FEV1

OCCUPATIONAL ASTHMA CAUSING AGENTS:

*List of Known Sensitizers as of 6/5/97**

Organic Chemicals

Acrylates

Methyl methacrylate, cyanoacrylates
Ethylcyanoacrylate ester
Plexiglass

Alcohols

Furfuryl alcohol (furan based resin)
Alkylarul polyether alcohol, polypropylene glycol
(combination)

Aldehydes

Formaldehyde
Glutaraldehyde
Urea formaldehyde

Aliphatic Amines:

Ethylene diamine
Hexamethylene tetramine
Triethylene tetramine

Aliphatic Amines: Ethanolamines

Monoethanolamine
Aminoethylethanolamine
Dimethylethanolamine

Anhydrides

Phthalic anhydride
Trimellitic anhydride
Tetrachlorophthalic anhydride
Pyromellitic dianhydride
Methyl tetrahydrophthalic anhydride
Himic anhydride

Amines, Aliphatic: Other

3-(Dimethylamino)-propylamine

Amines, Heterocyclic

Piperazine hydrochloride
N-methylmorpholine

Amines: Other

Chloramine T

Aromatic Hydrocarbons, NOS

Styrene

Azo Compounds

Azodicarbonamide
Diazonium salt
Azobisformamide

Chlorinated Compounds

Chlorhexidine

Fluorinated Compounds

Freon

Isocyanates

Toluene Diisocyanate
Diphenylmethane diisocyanate
1,5 Naphthylene diisocyanate
Isophorone diisocyanate
TDI, MDI, HDI, PPI (combination)
TDI, MDI, HDI (combination)
TDI, MDI (combination)

Phenols

Hexachlorophene

Polymers

Latex, synthetic
Polyvinyl chloride (fumes or powder)

Sulphonates

Iso-nonanyl oxybenzene sulphonate

Inorganic Chemicals

Metals

Aluminum
Chromium and Nickel (combination)
Cobalt and Nickel
Platinum
Nickel
Zinc fumes
Tungsten carbide
Chromium

Nonmetallic Elements

Fluorine

Miscellaneous Chemicals

Pharmaceuticals

Penicillins and Ampicillin
Penicillamine
Cephalosporins
Phenylglycine acid chloride
Psyllium
Methyl dopa
Spiramycin
Salbutamol intermediate
Amprolium
Tetracycline
Isonicotinic acid hydrazide
Hydralazine
Tylosin tartrate
Ipecacuanha
Cimetidine
Rose Hips

Dyes

Levafix brilliant yellow E36
Drimaren brilliant yellow K-3GL
Cibachrome brilliant scarlet 32
Drimaren brilliant blue K-BL
Persulphate salts and henna
Reactive dyes

Fluxes

Colophony
Zinc chloride, ammonium chloride (mixture)
Alkylarul polyether alcohol, polypropylene glycol
(combination)
Pylene glycol

Miscellaneous Chemicals, NOS

Tetraxzene
Oil mist

Biological Agents

Animal/Animal Materials

Laboratory animal
Egg protein (Egg producers)
Chicken
Pig
Frog
Lactoserum
Casein (cow's milk)
Bat guano

Fish/Fish Materials

Crab
Prawn
Hoya
Cuttle-fish
Trout
Shrimpmeal
Fish-feed, Echinodorus lava
Red soft coral

Insect/Insect Materials

Grain mite
Locust
Screw Worm Fly
Cricket
Bee moth
Moth
Butterfly
Mexican bean weevil
Fruit fly
Honeybee
L. Caesar larvae
Lesser mealworm, (Grain and poultry workers)
Fowl mite, (Poultry workers)
Barn mite, (Farmers)
Parasites (Flour Handlers)
Mites, (Flour Handlers)
Acarian, (Apple Growers)
Daphnia, (Fish food store)
Weeping Fig, (Plant Keepers)
Sheep Blowfly, (Technicians)

Biological Agents, con't

Larva of Silkworm

Plants/Plant Material

Grain dust
Wheat, Rye
Soya Flour
Lathyrus sativus
Vicia sativa
Buckwheat
Gluten
Coffee bean
Caster bean
Tea
Herbal Tea
Tobacco Leaf
Hops
Baby's Breath
Freesia
Paprika
Mushroom
Cacoon seed
Chicory
Sunflower
Garlic dust
Lycopodium
Sericin
Nacre dust
Henna

Vegetable Gums

Gum, Acacia
Gum, Tragacanth
Gum, Guar
Latex, natural rubber

Wood Dust or Bark

Western red cedar, (Thuja plicata)
California redwood, (Sequoia sempervirens)
Cedar of Lebanon, (Cedra Libani)
Cocabolla, (Dalbergia retusa)
Iroko, (Chlorophora excelsa)
Oak, (Quercus robur)
Mahogany, (Shoreal Sp)
Abiruana, (Pouteria)
African Maple, (Triplochiton scleroxylon)
Tanganyika aningre
Central American Walnut, (Juglans olanchana)
Kejaat, (Pterocarpus angolensis)
African zebrawood, (Microberlinia)
Ramin, (Gonystylus bancanus)
Quillaja bark
Fernambouc, (Caesalpinia echinata)
Ashwood, (Fraxinus americana)
Eastern red cedar, (Thuja occidentalis)
Ebony wood, (Disospyros crassiflora)
Kotibe wood, (Nesorgordonia papverifera)
Cinnamon, (Cinnamomum Zeylanicum)

Biologic Enzymes

B.subtilis
Trypsin
Papain
Pepsin
Pancealin
Flaviastase
Bromelin
Fungal amylase
Fungal amyloglucosidade
Fungal hemicellulase
Esperase

*Adapted from: Chan-Yeung M. Malo JL. Actiological Agents in Occupational Asthma. European Respiratory Journal. 1994. Vol.7. pp.346-371.

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***# PC20** = Provocative concentration to cause a 20% decline in FEV1*